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THE PARALYSES IN CHILDREN WHICH OC-
CUR DURING AND AFTER INFECTIOUS
DISEASES.*

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VARIOUS forms of paralysis occur during or after the well-known infectious diseases of childhood.

Pathologically, these may be cerebral, spinal, peripheral, or various combinations of cerebral, spinal, and peripheral disease. The cerebral cases are occasionally, but not often, due to embolism; more frequently they are the result of hemorrhage, the poison of the disease weakening the coats of the vessels. Strümpell¹ has suggested that a local encephalitis analogous to the poliomyelitis which produces the spinal form of infantile paralysis is sometimes the lesion. The spinal cases are usually myelitic, arising particularly from poliomyelitis or inflammation of the anterior horns; they may be recovered from or may be locally destructive and leave

* Most of the cases recorded in this paper have been studied by me at the service for Diseases of the Mind and Nervous System at the Philadelphia Polyclinic. A few of them have been taken from the record books of this service prior to my connection with the institution, and a few have been seen in private practice.

permanent paralysis. During recent years cases formerly supposed to be spinal, or even cerebral, have been found to be dependent upon local, diffused, or multiple neuritis; but only a few writers have discussed infantile and juvenile cases of this kind. Chapin² has written an article on peripheral neuritis and the painful paralyzes of early life, in which he directs attention to cases in his own practice and refers to a few reported by others. A few cases have also been recorded, and references to the subject are to be found in some of the text-books on nervous diseases and diseases of children. Most of the reported cases of neuritic palsies in children have come on during or after infectious diseases like scarlet fever, diphtheria, measles, whooping-cough, etc.; but some have been rheumatic, or, at least, apparently due to exposure. It will be seen from the records which follow that multiple neuritis, either alone or combined with myelitis, is of somewhat frequent occurrence, and that the palsies are sometimes of a peculiar and interesting type. The cases, eighteen in number, represent sequelæ of nearly all the infectious diseases of childhood; but some, as diphtheria and measles, largely predominated as antecedents.

Cases are first given as occurring after measles, scarlet fever, diphtheria, whooping-cough, and mumps; but besides these, the notes include a few after such diseases as influenza, malaria, and typhoid fever. After presenting the cases, diagnosis, prognosis, and treatment will be briefly considered; but the paper is intended to present a series of cases with their apparent teachings, rather than to discuss at length the subject or its literature.

CASE I.—MULTIPLE NEURITIS OCCURRING AFTER MEASLES AND WHOOPING-COUGH.—PROBABLE RACHITIC CURVATURE.—RECOVERY.

B., at the age of twenty months, about April 1, 1890, had measles, from which she recovered without sequelæ. In June she was taken with whooping-cough, and toward the last of this month was attacked with fever and diarrhœa, which lasted several days. Her legs became weak, and in a few days she could not walk, and was in great pain. She had walked for several months prior to the whooping-cough. When first examined, early in July, she was completely paralyzed in both lower

extremities, which were everywhere exquisitely painful to touch and movement; and attempts to examine her threw her into tremulous excitement. Her mother and nurse handled her with the utmost care, but in spite of this she would often scream. In various ways she was tested to determine that her fear and apparent suffering were not due to fright; cautiously manipulating or squeezing her feet or legs when she was not looking always caused cries and other evidences of pain. Her feet and legs dangled helplessly, but the upper part of her body was totally unaffected. It was impossible at first, because of her suffering and fright, to examine her with electricity.

The diagnosis was made of multiple neuritis following measles and whooping-cough, and the child was directed to be kept as quiet as possible all the time. Applications of hot water were made to her legs. At first she was given small doses of salicylate and bromide of sodium, and a little later minute doses of bichloride of mercury; she was also carefully and fully nourished. For two or three weeks her suffering was extreme, and, although slow, gradual improvement took place, the pain and tenderness did not wholly disappear until the latter part of August, last from the upper part of the thighs. In September her limbs showed general wasting. Treatment with galvanism was begun about September 15th. Here and there a muscle would not respond thoroughly to the galvanic or faradic current, but no advanced electrical changes were present. After the pain had entirely disappeared from her extremities, she was unable to use her limbs; but from this time she slowly recovered power. She was quite thin, and still had a cough. She was placed upon reduced iron, cod-liver oil, and Lugol's solution, alternating the latter for a time with Fowler's solution and the compound syrup of the hypophosphites. The use of galvanism three times a week was continued, and massage, with cod-liver oil, was used on alternate days.

In October she was strong enough to stand and make a few steps without assistance; but it was now found that she was unable to support her trunk properly, and seemed to have some pain in her back. A slight prominence was found in the middle lumbar region, which was thought to be a rachitic curvature, or threatened caries. She was now placed on her back; a slight frame-work was placed under her mattress, so that she could be taken out-of-doors without changing her from a recumbent position. Massage, electricity, and tonics were continued. The child thrived under this treatment, but she was not allowed to get up for about three months, when she was fitted with a light leather jacket. This was only kept on for a few hours at a time at first, and only when she was up and about. Later, however, she wore the jacket all day, but was allowed to take it off at night. In six months the jacket was taken off, at first for a few hours at a time and afterward for a longer period, until it was left off altogether. The father of this patient when a small child had, or was threatened with, some spinal trouble, probably caries, for which he was kept flat on his back for several weeks.

Both measles and whooping-cough preceded the development of paralysis in this patient; the two infectious diseases occurring closely together, as often happens. The following are brief notes of cases in which measles alone preceded some different forms of paralysis in children:

CASE II.—LOSS OF SPEECH AND BOTH SENSORY AND MOTOR PALSIES AFTER MEASLES.

Unfortunately the notes of the first of these cases are not complete. The patient came, off and on, to the Polyclinic Dispensary for Nervous Diseases for about three months.

S. W., aged three years, eight weeks before coming under observation had measles. The eruption was only imperfectly developed. During the attack, three days after its commencement, she lost speech, and also almost completely the senses of touch, smell, taste, sight, and hearing. Diminution of the various forms of sensibility was still present when she came to the service, and the muscles of the neck were so paralyzed that she could not hold her head firmly in any position, but allowed it constantly to drop forward; she also had paralysis of the legs and arms. The first signs of improvement were observed two or three weeks after the beginning of the measles. Galvanism and massage, with strychnine and iron internally, were employed, and the patient gradually recovered her sensory powers, and also from the paralysis; but at the end of three months it was noted that the patient, while able to walk better, was still weak, and complained of pain in the soles of her feet.

CASE III.—MEASLES FOLLOWED BY BRONCHITIS AND THIS BY PARTIAL PARALYSIS.

L. T., aged five years, four or five months before he was first examined, had an attack of measles, but entirely recovered, and remained so until six or seven weeks before coming under observation, when he developed bronchitis with some cough and nasal catarrh. His sister was affected in the same way. His mother noticed after this that he had regurgitation of food, water, etc., and also that he dragged his left leg in walking. The boy's chest was filled with moist and dry râles, and he had a large tumid stomach. Although he dragged the left leg, the limb was not atrophied. He had nocturnal incontinence of urine. At one time, for a short period, he was unable to hold up his head. He was placed upon strychnine, massage, and electricity, and made some improvement, but came to the service only a few times, and then ceased attendance.

CASE IV.—DIPLEGIA AND OCULO-MOTOR PARALYSIS AFTER MEASLES.

This patient, a girl of four years, came to the service in October, 1887. She had had measles in May, and since then had not been able to walk, and could talk but little. The attack of measles was very

severe, the child being sick about two months. While still in bed she lost power in both her lower and upper limbs; she also had convergent strabismus in the right eye. Both knee-jerks were absent, and the limbs were somewhat wasted. The patient continued to come for treatment for more than two months, steadily improving under galvanism, massage and tonics.

I have among my notes the record of a case of measles which was followed by complete aphonia, which lasted several weeks, and which was not accompanied by any other paralytic symptoms.

Although the notes of these four cases, except perhaps of the first, are meagre, they are of value for the discussion of the pathology and treatment of such affections.

Allyn^s collected forty-one cases of paralysis occurring during or following measles, including one of his own. Thirty-five of these, according to his analysis, were cerebral in origin; but it is doubtful whether this indicated the true proportion. Probably many cases of spinal and peripheral palsies have not been recorded. Allyn does not refer to cases of multiple neuritis after measles, but describes several of spinal origin, some of which may just as well have been peripheral, or both spinal and peripheral. One case of Liegard's, quoted by him, may have been in whole or part peripheral. "Between five and six days after the attack of measles began palsy was first noticed by the mother. Nine days later the condition was as follows: In addition to great weakness of the upper and lower extremities, the head fell forward on the chest from palsy of the muscles of the back of the neck. This manifestation had been gradually increasing for two days. Deglutition at the same time became more and more difficult; mastication was also interfered with. The skin appeared to be insensitive to puncture and to pinching." In less than three months the patient fully recovered.

While so large a proportion of Allyn's cases were of cerebral origin, none of the four cases here reported corresponded with any ordinary types of cerebral palsy.

The first was distinctly one of multiple neuritis, with probably also some rachitis or osteitis. In the second, in which common and special sensibility, as well as motor power and speech, were somewhat affected; and in the other two cases, in which the nerves and muscles of the limbs, throat, and eyes were attacked, the best explanation is one which makes the symptoms dependent upon some disease of the nerves or their nuclei. Commonly these cases, whether after measles, diphtheria, or other infectious diseases, are regarded as forms of neuritis or neuro-myelitis; but they differ from neuritis of the clearly recognizable type in the absence of pain and hyperæsthesia. The senses everywhere were blunted, and the muscles paralyzed, or partially paralyzed in one of the cases, and yet the patient, however uncomfortable, did not suffer directly from pain. A better explanation of such cases is perhaps that which has been advanced by Thompson⁴ and others, more particularly with reference to the nervous symptoms and sequelæ of diphtheria, namely, that ptomaines are formed as the result of the changes which the infection produces; and that nerve and muscle affections are the result of toxæmia or poisoning, which causes some degeneration of tissues, but not a true inflammation. These remarks are applicable to all the cases which will be recorded in connection with infectious diseases.

Since the publication of Allyn's paper another case of paralysis following measles has been reported by Carpenter.⁵ The patient, three years old, had mild measles, which, however, had been preceded by a scarlatinal affection, also mild. On the third day the child had an attack of stupor with high fever, but in a few days was convalescent, and was then found to be partially paralyzed or inco-ordinate in the right lower limb. Carpenter thought it necessary to make a concealed post-nasal diphtheria responsible for these disturbances; but I do not see why, as the cases here reported, and many others, show that the infection of measles may produce the same paralytic results as that of diphtheria. A case like

this would probably generally be regarded as cerebral because of its one-sided character; but the theory of toxæmia and the production of ptomaines would make it equally easy to explain it as peripheral, nuclear, or cerebral. The fact that the child recovered under strychnine in ascending doses would be in favor of its peripheral, or, at any rate, its toxæmic nature.

PARALYSES FOLLOWING SCARLET FEVER.

The next two cases are examples of paralysis following scarlet fever. They are interesting as showing that neuritis was undoubtedly present in both, although the first the palsy that was left was of the hemiplegic type.

CASE V.—MULTIPLE OR DIFFUSED NEURITIS WITH SUBSEQUENT PARALYSIS OF RIGHT ARM AND LEG FOLLOWING SCARLET FEVER.—RECOVERY.

E. S., aged six years, in July had scarlet fever, with which she was very sick for two weeks, and during this time complained of intense pain in the legs and arms. At the end of four weeks, on attempting to walk, it was found that the right leg was weak, and that she was unable to get her foot to the floor on account of the pain the effort caused. Any attempt to extend or handle the foot would bring on intense pain. At the same time she had paralysis of the right arm which was also sensitive. The pain on handling gradually disappeared, and she regained to some extent the power in the leg. When first examined, in November, her right arm and leg were still weak; she could only use the arm when compelled to, and in walking dragged the right foot. The muscles responded to both currents, but with quantitative changes in both arm and leg. Speech was slightly affected. She improved very rapidly under digitalis, strychnine, tonics, and galvanic treatment, and at the end of two months, from November, had regained the complete use of both arm and leg.

This patient had a marked mitral regurgitant murmur, the natural inference from which would be that the right-sided paralysis, or paresis, was due to embolism; but the history of improvement and recovery would not tend to confirm this view. Because a child with scarlet fever, or any other serious infectious disease, develops an endocarditis and valvular murmur, it does not necessarily follow that the paralysis, even if it is one-sided is

cerebral. The same toxic or infectious agency may set up both an endocarditis, and either a limited or widespread neuritis, as this case and the one to be detailed next would seem to show.

Most authorities hold to the view that monoplegic and hemiplegic palsies following the exanthemata are due to local cerebral lesions. Abercrombie,⁶ for instance, discusses several views, most of which have been alluded to at the beginning of the paper, to account for these cases, but does not refer to multiple or diffused neuritis, or to toxæmia of the nerves and nerve centres. He refers, first, to the theory of Strümpell of a localized polioencephalitis; secondly, to that of hemorrhage, strongly advocated by Eustace Smith, and also by Henoch and Barlow; thirdly, to Goodhart's views of meningeal hemorrhage or inflammation; fourthly, to the theory of thrombosis of the veins and sinuses supported by Gowers; fifthly and lastly, to that of embolism with softening, in explanation of which he cites the authority of Ross and Goodhart, and to which he gives his own adhesion, saying, that in all probability embolism of one of the middle cerebral arteries is the cause of the paralysis that supervenes during convalescence from the acute infectious disorders.

This patient having a positive and persistent cardiac murmur, embolism would be first thought of; but the general pain and sensitiveness which disappeared under time and treatment, and the eventual complete recovery is, as has been stated, against this view, although partial recoveries do occur in cases of embolism.

The next case would seem also to point to the neuritic origin of at least some of the cases of paralysis following scarlet fever. This patient was for several months at the Polyclinic under the care of Prof. H. Augustus Wilson, of the Department of Orthopædic Surgery, having come into his ward because of deformities associated with the paralysis. I am indebted to Dr. Wilson for the opportunity to use the case.

CASE VII.—PARALYSIS OF THE UPPER AND LOWER EXTREMITIES, WITH ATROPHY AND DEFORMITIES AFTER SCARLET FEVER.—RECOVERY OF THE UPPER EXTREMITIES, BUT PERSISTENCE OF PARALYSIS AND ATROPHY IN THE LOWER.

J. D., aged ten years, had always been well until about a year before coming to the Polyclinic, when he had scarlet fever, three days after the beginning of which he had an attack called inflammatory rheumatism, but one which probably could have been more correctly described as multiple neuritis with arthritis and endocarditis. He developed both mitral stenosis and aortic regurgitant murmur. His first complaint was that his feet pained and burned, and later the pain and burning spread to all parts of his legs and then to his arms. In a week he had pain and extreme sensitiveness in the four limbs, in the trunk, and even in the face and head. His sufferings were by no means confined to the joints. The bed clothes had to be propped up so that they would not touch him. His knees were a little swollen for a time, but swelling of the joints was not a marked symptom. He lay helpless for two months, so far as his lower extremities were concerned, unable to move a toe. During this time his arms were also painful and helpless, but not so much as his legs. His condition varied from time to time; both arms were affected; but sometimes one was more painful, helpless, and contracted than the other. For six weeks his head was drawn downward toward one shoulder.

His arms slowly got well. After two months he began to use his left foot and leg, putting it out, kicking, etc., but ten days later he again lost this power after catching cold. The pain came back with renewed force, and was now confined to his lower extremities. In about ten weeks from the time of his first attack his mother noticed that his legs were drawing upward; and this contracturing increased until they assumed a position of extreme deformity, the thighs bent on the pelvis and the legs on the thighs. Wasting of the limbs was not a marked symptom until five months after the first attack. About four months after the first attack he developed bed-sores, one near each trochanter. In July a central ischiatic eschar developed, and this became very large. He never lost control of his bladder and bowels. Electrical examination showed that the muscles above the knees responded to both currents, although the response was depressed. The muscles below the knees did not respond to either current, although they were carefully tested. The patient's present condition might be briefly described as one of nearly complete paraplegia, with atrophy, and degeneration reactions in many muscles, and with also marked contracture deformities.

The case was certainly one of great interest and importance. In the first place, although marked endocarditis occurred and murmurs both mitral and aortic were present, the terrible paralytic condition of the patient was

not of the cerebral embolic type. We may have double hemiplegias or irregular palsies on both sides of the body from embolism; but cerebral paralysees do not give marked atrophies and changed electrical reactions, with a history of pain and hyperæsthesia, sacral eschars, etc. The atrophic paralysis was in all probability the result of an inflammatory affection which attacked both nerves and spinal cord, and was due to the action of the infection upon nerves and nerve centres.

The next case had three separate attacks of paralysis before she was eighteen years old, the first two succeeding acute eruptive diseases, and the last being apparently of spontaneous origin.

CASE VIII.—PARALYSIS AND APHASIA AFTER AN ERUPTIVE AFFECTION AT THE AGE OF EIGHTEEN MONTHS.—A SECOND ATTACK OF PARALYSIS AND APHASIA AFTER SCARLET FEVER WHEN NINE YEARS OLD.—FACIAL PARALYSIS OF UNKNOWN CAUSE AT THE AGE OF EIGHTEEN YEARS.

M. A., when eighteen months old, had a fever and an eruption, which the mother said was something like hives. A few days after the beginning of the attack she was found to be completely paralyzed and speechless; she could not raise her head or lift her arms. In six weeks she got so well that she could creep, and continued to regain power very slowly; but her speech did not return for seven months. In the course of a year she was perfectly well, and remained so until she was nine years old, when she had scarlet fever with severe sore throat. Her temperature was high, and with the scarlet eruption here and there were spots like blisters. The day after the appearance of the rash she became speechless and completely helpless. She soon began to walk again, and in a few weeks recovered from the paralysis; but for two years she did not talk. She remained well until five days before coming to the Poly-clinic, being then about eighteen years old, when she began to complain of pains in the eyes and back of the head on the left side, and the next morning it was noticed that her face was twisted to the opposite side. Ever since she had had pain in her left eye. On coming to the clinic she had complete paralysis of the left side of the face, but sensation and taste were not affected. The case was treated for some time at the dispensary, and she got nearly well; it was one of the forms of Bell's palsy, the lesion being probably outside of the Fallopiian aqueduct. She had for a time degeneration reactions.

Such a case indicates a peculiar vulnerability of the peripheral nerves in some individuals, so that infectious disease, exposure, or any of the numerous causes of peripheral palsies are likely to be unusually effective.

PARALYSIS AFTER VARIOLA.

No case of juvenile paralysis following variola has come under my observation, although Gowers says that spinal symptoms are more common in connection with small-pox than with any other of the eruptive fevers. Except when epidemic, small-pox is, of course, a rare disease, even in large cities like Philadelphia, and for this reason its nervous sequelæ are seldom observed in dispensary and private practice. From analogy we would expect to find forms of organic nervous disease the same or similar to those met with after other contagious or infectious disorders. We find reported by various observers such sequelæ as ocular paralysis, atrophic paralysis of the legs and arms, ataxia, acute ascending paralysis, and aphasia. Among the lesions which have been demonstrated are disseminated myelitis, poliomyelitis, and acute degeneration of the nerves. Welch⁷ speaks of having seen two or three cases of aphasia as sequelæ of small-pox, and believes that this affection may be the result of a circumscribed encephalitis. According to his experience, recovery from this speech-defect is slow. No distinction is made by the authors I have consulted as to whether the post-variolous affections occurred in adults or in children; probably they were usually observed in adults. The same pathological agencies are, however, at least as likely to affect the susceptible nerves and nerve centres of youthful patients as those of adults, if the former are unfortunate to suffer from small-pox.

DIPHThERITIC PARALYSIS.

Diphtheria has probably contributed more cases of paralysis in children than any other infectious disease, possibly more than all of them put together. I have seen a number of such cases, but will include in my series only a few which have presented points of special interest. The first is one of unusual type—a neuro-myelitis—a true multiple neuritis concurring with a poliomyelitis, and while the neuritis was recovered from,

the myelitis left the child with a form of permanent and serious spinal paralysis. The resulting condition, in other words, presented the usual features of a case of spinal infantile paralysis occurring independently of any of the ordinary infectious diseases.

CASE IX.—MULTIPLE NEURITIS AND POLIOMYELITIS AFTER DIPHTHERIA.—RECOVERY FROM THE NEURITIS, BUT PERSISTENCE OF A SERIOUS TYPE OF SPINAL PARALYSIS.

M. H., aged four years, was well until eleven months before she was first seen, when she began to complain of headache and sore throat. The pain was most severe at the nape of the neck. She also had some fever, and her throat was sore for three days. Her brother had just had an attack of sore throat, and diphtheria was in the neighborhood. In less than twenty-four hours she lost the use of her lower limbs. The fever continued three weeks, and all this time she had pain in her neck. She was completely paralyzed, with the exception of her arms, and was hyperæsthetic over almost the entire body below the arms, the slightest touching or handling causing intense pain. The parents were certain that the child could use her arms during the attack, but were not certain about their sensitiveness. She was at no time unconscious, and never had involuntary evacuations from the bowels or incontinence of urine.

After three weeks she commenced to improve slowly, but at times would have a slight relapse, and it was fully three months before she could sit up alone. At the end of six months, pain and hyperæsthesia had disappeared, and she gradually regained power in her lower extremities; but for three months before coming for examination she had remained in about the same condition. The entire left lower extremity showed wasting, and nearly all the movements of the left leg were abolished. In creeping she partially flexed this leg by some of the thigh muscles. The leg was not flushed, blue, or cold. None of the muscles of the left leg responded to either faradism or galvanism. In the right thigh a few of the muscles were also paralyzed. She was seen but once eleven months after the attack, and improvement had been practically at a stand-still for two to three months.

Paralysis of the ocular muscles would seem to be neither very common nor very rare, and I have brief notes of two of these cases in which an affection of the oculo-motor nerve was associated with other palsies.

CASE X.—OCULO-MOTOR AND PALATAL PARALYSIS, AND PARESIS OF THE LIMBS, FOLLOWING DIPHTHERIA.

F. K., aged eleven years, colored, had diphtheria three months before coming under observation—a very severe attack which kept her in bed for

two weeks. She had some paralysis of the palate, with regurgitation, etc., and shortly before she began to go about the house she lost her voice. She also had internal strabismus of the right eye. The grasp of both hands was feeble, and she had partial loss of power in the arms, the deltoid and other shoulder muscles being distinctly affected. In walking, her gait was slow and feeble; she could kick, but could scarcely raise her heel from the floor. At the time of the examination the electrical responses were present. Under faradism and strychnine she improved rapidly.

CASE XI.—OCULO-MOTOR AND PALATAL PARALYSIS, AND
PARAPLEGIA, AFTER DIPHTHERIA, WITH ABOLISHED
KNEE-JERKS.

J. T., aged three years, white, about two months before coming under observation had diphtheria. A sister and brother died of the disease during his illness. Two days after he got on his feet it was noticed that he had slight stiffness in the right leg in walking, and complained of pain in his head. At night he had screaming spells, and also occasionally vomited. In a week his mother noticed that he was paralyzed in both legs, and a few days after this that he had convergent strabismus. He had slight regurgitation and convulsive cough on trying to swallow. The knee-jerks were abolished. He had no history of pain or sensitiveness in the limbs. He was treated for a short time, with improvement, and disappeared from the clinic.

The occurrence of various types of paralysis at comparatively long periods after diphtheria, and other infectious diseases, has often been noted. Ferguson⁹ records a case which began four weeks after complete recovery from the measles, and advanced until the tibialis anticus, the extensor longus digitorum, the peroneus tertius, and the extensor proprius pollicis were almost completely atrophied, and showed extreme reactions of degeneration. In six months from the onset of the paralysis there was a fair amount of recovery; but the calf muscles did not find sufficient opposition and underwent some contraction. Ferguson⁹ holds that this case points clearly to the view that such paralyses are due to some poison left in the system by the disease, a poison which acts after the disease itself has disappeared. "We could hardly suppose," he says, "any of the real *materia morbi* of the disease still existing in the system four weeks after the disease itself had disappeared, and desquamation had been completed, and at a date when

no one would regard the person as still carrying any contagion. . . . It is only fair to suppose that the paralysis was due to some agency or poison that was left in the system by the measles."

CASE XII.—PARALYSIS OF THE LEG ONE YEAR AFTER DIPHTHERIA, WITH RECOVERY.—PHARYNGEAL PARESIS AND TORTICOLLIS ONE YEAR LATER.

In this case the patient was not paralyzed until a year after recovery from diphtheria, and two years later had a second localized attack of neuritis, causing a tonic spasmodic affection.

C. M., aged five years, colored, two years before coming to the Polyclinic had diphtheria, with convulsions, at the beginning of the attack. One year afterward he began to lose power in his legs from the knee down, with pain in the right knee-joint. About one year after this, and three weeks before coming under observation, he took cold, and a week later had torticollis, the head moving to right. He had some difficulty in deglutition before the torticollis appeared. At the same time that he began to complain of his neck he complained also of pain in his right arm, which disappeared on rubbing with liniment several times. Tetanic contraction was present in the trapezius muscle as well as in the muscles under it.

It may be questioned whether the relation of cause and effect existed between the diphtheria and the nerve affection in this case; nevertheless, a profound toxæmia might have so affected the nervous system of the patient as to have rendered him liable to such inflammatory and paralytic attacks.

CASE XIII.—SLIGHT PALATAL PARALYSIS AFTER DIPHTHERIA, WITH RENEWED TROUBLE WITH THE THROAT AND RECURRENCE OF THE PARALYSIS.

W. E., white, aged eleven years, school-boy, had diphtheria lasting three weeks. He was sent to the country, and appeared to be well, but in three weeks had symptoms of paralysis of the throat. Under treatment he almost entirely recovered, and was comparatively well when his throat began to trouble him again, and he developed nasal voice and regurgitation of food, but had no definite paralysis of the limbs, or any other groups of muscles than those of the pharynx. He was, however, very weak and easily fatigued.

These five cases of diphtheritic or post-diphtheritic paralysis, with the exception of the first, present in their symptomatology no unusual features. The first, with its

persisting palsies, presents the distinctive features of a poliomyelitis. Cases like the others detailed might have been given in considerable numbers, showing various combinations of ocular, palatal, pharyngeal, and limb palsies, with abolished knee-jerk. The common type of diphtheritic parylysis is illustrated by these cases of multiple, or, perhaps what might be better designated, irregularly distributed paralysis. Sensory symptoms, while sometimes present, were not of a decided character, although to a certain extent these may have disappeared before the cases came under observation.

Commonly these cases have been regarded as instances of either neuritis or myelitis, or a combination of these pathological conditions; but even a superficial study of their symptoms shows that this explanation is doubtful. The absence of pain and hyperæsthesia does not accord with the view that an active neuritis has been present, and destructive myelitis is shut out by the history of recovery. Doubtless more or less degeneration of nerve and muscle, and sometimes also of the spinal cord, takes place in these cases, and disappears under reparative processes in the lapse of time and as the result of treatment; but this degeneration when present is probably not secondary to active inflammation, but the more direct result of some other process. Bacteriological investigations and speculations throw some light upon the nature of these cases, and, as indicated in several places in this paper, upon the nature in general of the organic changes which occur during or after infectious diseases. Micrococci and bacteria, of known species, and unknown bodies, have been found in the vessels, and in the nerve sheaths and fibres and centres, by various observers. Inflammation may ensue from the presence of these organisms; or the tissues may degenerate; or the functions of the nerves and nerve centres may be inhibited because of their presence.

With reference to these questions Gowers makes the following significant remarks: "Are these changes the result of some nerve poison left behind by the diph-

theritic virus, a poison which multiplies in the body without causing general symptoms, and is certainly capable of infecting another person? Or are they due to the diphtheritic poison, bacterial or other, which acts on the nerve elements during the primary disease, although the effect on the nutrition of the cells and fibres does not show itself until some time after the virus has ceased its active ravages? Or are they due to some poison associated with that which causes the throat affection, of a similar nature, but not necessarily proportioned to the latter—indeed, not necessarily co-existing with it—and producing its effects without pyrexia? To these questions at present no answer can be given. But it may be noted that the absence of any relation between the intensity of the diphtheria and of the subsequent paralysis accords best with the last theory, and this is supported, also, by a very remarkable series of facts recorded by Boissarie. In a certain district of Paris there occurred a series of cases of severe diphtheria, and at the same time a series of cases of paralysis of the palate, eyes, heart, etc., perfectly like that which occurs after diphtheria, and accompanied by albuminuria. The remarkable fact is that in these cases of primary palsy there was no history of preceding sore throat, and in several of the cases distinct diphtheria followed the paralysis, which lessened during the throat affection. Some of the cases of primary palsy seemed to arise distinctly by infection.”

The explanation of the paralytic and other phenomena, which attributes them to the primary or secondary action of the diphtheritic poison would seem most satisfactorily to account for the conditions; that which assigns a large part to ptomaines generated by the diphtheritic poison has most in its favor. Prognosis and treatment will vary according to the measure which the physician takes of the extent of the primary and secondary changes.

In considering paralysis in children in general, and diphtheritic paralysis in particular, it is a practical point of great importance to remember that occasionally the paralytic affections are due to some infectious agency,

even when no history of previous infectious disease can be discovered, as, for instance, in the Paris cases cited from Gowers, in which the paralysis occurred in a series of cases in which the patients had no preceding histories of sore throat. It somewhat frequently happens that cases of paralysis are seen in children during an epidemic of scarlet fever, diphtheria and measles, the children affected with the paralysis, however, apparently not having suffered from the infectious disease.

PARALYSES FOLLOWING WHOOPING-COUGH.

Whooping-cough, while less frequently than most other acute infectious diseases of childhood, the forerunner of paralysis sometimes precedes its cerebral, spinal, or peripheral types. It will be remembered that the first case recorded in this paper occurred after both measles and whooping-cough. The following are brief notes of one case of this kind.

CASE XIV.—NEURITIC PARALYSIS FOLLOWING WHOOPING-COUGH.

F. S., fifteen months old, was perfectly well until five weeks before she came to the service, when she had a severe attack of whooping-cough, and three days before coming to the clinic her mother noticed she could no longer use her right leg. She had never walked, but was in the habit of creeping and using her legs. When the limb was handled she gave evidence of great pain; she also had marked pain in the right hip-joint and in the back. She remained in this condition for nearly two weeks when the pain largely disappeared, but the limb was left paralyzed. Knee-jerk was absent on the right, but present on the left side. She came for treatment only three or four times. When last seen, the paralysis had improved, but some still remained. The muscles did not lose their response to electricity, and the case was probably one of a form of mild neuritis associated with rachitis.

PARALYSIS AFTER MUMPS.

Reports of cases of paralysis occurring after mumps are exceedingly rare. Boas, according to Gowers, has recorded an instance of palsy of accommodation which was met with in only one case in an epidemic. The paralysis affected all four limbs, the legs first. Lan-

cinating pains preceded the paralysis; the deep reflexes were abolished; the muscles gave reactions of degeneration, and slight pressure caused pain. The paralysis continued for four months and got well under iodide of potassium. Gowers suggests that in each of these cases undiscovered diphtheria may have been present. In Joffroy's¹⁰ case slight angina was observed.

CASE XV.—PARALYSIS OF THE LEFT LEG FOLLOWING SWELLING OF THE RIGHT PAROTID GLAND.—PERSISTENCE OF THE PARALYSIS.—PROBABLY BOTH NEURITIS AND POLIO-MYELITIS.

C. B., aged five years, in July had a swelling of the right parotid gland, which extended in front and around the ear. She had no fever, and did not complain of pain; the swelling lasted twenty-four hours, and, as it was passing off, it was found she was unable to use the leg, which was cold and very sensitive to touch. The mother said the sensitiveness extended from the hip downward, but was more marked below the knee than above it. This condition gradually disappeared, and when seen in September, the hyperæsthesia was entirely gone, but most of the muscles below the knee were paralyzed and atrophied, and to a less degree those above. She had a recurved knee. She could pull her heel up and bend her toes partly under, but could neither dorsal flex, abduct or adduct the foot. The muscles most affected were the anterior tibial, the extensor proprius pollicis, and the extensor longus digitorum. She could feebly flex the thigh on the leg, the sartorius and a portion of the quadriceps extensor being paralyzed. She walked with a flat everted foot. Knee-jerk was absent, and degeneration reactions were present.

This case presents the typical features of a spinal infantile paralysis, and the parotid swelling may have been a coincidence. Under several months' treatment the paralysis was but slightly improved.

PARALYSIS DURING THE INFLUENZA.

During and following the influenza many forms of nervous diseases, functional and organic, have occurred, and not a few of these have resulted in paralysis of some type. Neuritis, myelitis, cerebritis, meningitis, and combinations of these inflammations have occurred, but I do not know of any case in children, resulting in paralysis, which have been reported, although such are probably on record.

At the height of the recent epidemic of influenza I saw a case which presented the features of a multiple neuritis, at first attacking both legs, but after several weeks the patient, a boy, six years old, was left with paralysis of the anterior tibial and extensor proprius pollicis of the left leg. The case in all its features corresponded to a localized poliomyelitis. An unusual number of cases of juvenile paralysis were brought to the Polyclinic during the declining months of the epidemic of influenza, but whether the epidemic influence had anything to do with their production is uncertain.

MALARIAL PARALYSIS IN CHILDREN.

Some interesting observations have been made on multiple neuritis in children, the result of malarial poison, by Gowers, Chapin, and Browning.¹¹ Chapin¹² refers to three such cases, one in a boy, thirteen years old; another in a boy, aged five years and six months; and a third in a girl, nine years of age, all of which recovered under anti-malarial treatment. The limbs of all these cases were painful, and paraplegia and paraparesis were present. Chapin believes that in cases of paralysis in children, in which pain has been a prominent and persistent symptom, where loss of power has perhaps been gradual and ascending, and where there has been slow but complete recovery, but without deformity, a careful investigation will generally reveal a peripheral lesion the cause of the trouble. Browning also gives several cases presumably of malarial origin, and discusses the nerve complications and sequelæ of malaria. He records various observations on neuritic malarial paraplegias: "The main complaints from which they are brought is that the legs have become weak and paralyzed. The children have been too young and uncommunicative to say whether it was tenderness and pain that interfered with their walking. They object to making attempts, and when stood up remain where placed with feet somewhat apart, or make clumsy at-

tempts at progress and drop down helplessly." I have seen one case of paralysis of the arm, leg, and face which may have been of malarial origin; there was, at least preceding the attack, a history of chills and fever of the tertian type.

PARALYSIS AND LOSS OF SPEECH DURING AND AFTER
TYPHOID FEVER.

Typhoid fever in children as well as in adults is sometimes followed by paralysis. At the meeting of the Association of American Physicians in Washington, in September, 1888, Ross,¹³ of Montreal, read a paper on Some Forms of Paralysis after Typhoid Fever. In the discussion of this paper many interesting facts and views were elicited. Starr thought that while neuritis and paralysis were often due to infectious causes, the former was an infrequent sequel of typhoid fever. Osler and Wood believe that typhoid fever might light up either a neuritis or a myelitis; and Delafield that the poison of the severe infectious diseases caused acute degeneration to which might or might not be added inflammatory changes. Ross showed that while typhoid fever was a great strain on the nervous system, causing general exhaustion and anæmia, in addition, it produced sometimes nervous lesions which consisted usually in interstitial neuritis, but also sometimes in parenchymatous degenerations of the spinal cells and cerebral cortex, the results being recoverable paralysis, paraplegia, and sometimes loss of sensation. They were undoubtedly caused by the typhoid poison, not by the fever, exhaustion, or anæmia. The nervous phenomena were all most invariably both motor and sensory, and mainly the nervous injury was manifested by pains, areas of increased cutaneous sensibility, and paralysis. Ord, in the discussion of the paper of Ross, spoke of loss of speech during typhoid fever. He had seen cases after severe attacks, in which patients had spoken at the rate of a word every two or three seconds; he had also seen a patient come out of a condition of uneasiness, be unable to speak at all, and recover the power of

speech only after a few days. In one case he found the patient not only unable to speak at first, but that he had also lost the faculty of remembering words, and had to learn to speak over again. This was more likely to occur in young children from seven to eight years of age; in one case the process of learning to speak had to be recommenced and developed as originally. He thought that the changes were in the central nervous system rather than in the periphery, that there were inflammatory changes penetrating the tissues.

As occurring during typhoid fever, Gowers mentions first, sudden hemiplegias, the result of vascular lesions, which may either persist or pass away, according to the position of the lesion; in children especially, convulsions usually attend the onset of these cases. Besides these cases the most common nerve symptoms indicate a special influence of the poison on the brain; among these are deafness and loss of power of speech. According to Gowers, there is generally complete speechlessness, and although it is called aphasia, it is not a disorder of speech such as occurs in organic disease of the brain. In one case under his observation the condition developed gradually by disuse of the lips, which were dry and cracked; the loss of the labial articulation rendered the words almost unintelligible, and they gradually ceased to be uttered. The condition may last for some weeks, and in one recorded case it was associated with general choreic movements.

Dr. C. S. Turnbull has kindly furnished me with the notes of the following case:

CASE XVI.—HEMIPLEGIA DURING THE COURSE OF TYPHOID FEVER, AND LOSS OF SPEECH DURING CONVALESCENCE.
—RECOVERY.

In April, 1889, E., nine years of age, was suddenly attacked with high fever. In two days she had petechia, followed by epistaxis and headache, but she had no abdominal symptoms except gurgling at the iliac fossa. The attack lasted three weeks and was characterized by subnormal temperature in the early morning. Other symptoms were not marked.

In April, 1891, just two years later, she was attacked as suddenly, in the midst of apparent health, with fever, and in forty-eight hours her temperature was 102.2° F., with muttering delirium; tympanites was marked and alarming. She had some loose passages, but no typhoid stools until after the first week of her illness. Grave pneumonia succeeded, and the attending physician feared she would succumb to lung choking, but he used oxygen with good effect, also stimulants and muriatic acid. Her temperature ranged high, 104°, or even more. Ice was kept on her head constantly for two weeks. She had but little ability to swallow and was markedly deaf, but responded to a loud voice; she had no middle ear complications.

After the muttering delirium ceased, about the middle of the second week, the patient made no sound except now and then a cry as if in pain; soon after she smiled, made a little noise, and followed with her eyes persons about the room, and the movements of the lips. The paralysis gradually passed off about the sixth week, and one Sunday, when alone with the nurse, she said "pug dog," which were the very last coherent words she spoke when nurse No. 2 was brought in on the third day of her illness. Nurse No. 1 said: "E., who is coming up-stairs?" E. said "pug dog," and laughed.

About the third week partial paralysis of the left arm and shoulder was noticed. Massage and friction were employed. Deafness occurred with the partial paralysis, but did not remain after the third week, after which also consciousness of her surroundings returned.

In the first attack she was attended by Dr. J. C. Wilson, who pronounced the affection surely typhoid fever. In the second attack, Dr. Wilson was consulting physician, and said the case was decidedly a high temperature typhoid, as the first had been a low temperature. At the present time, May, 1892, no nervous symptoms remain, except at times a little hesitancy in speaking; that is, when telling a story, or when perhaps embarrassed by listeners.

CASE XVII.—LOSS OF SPEECH DURING THE COURSE OF TYPHOID FEVER.

I have had the opportunity of seeing one case of aphasia, or, of at least, of the loss of speech and the ability to recognize spoken words, which occurred during the progress of typhoid fever. This patient was a child, five years old, who, in the second week of a severe case of typhoid fever, ceased to talk for eleven days. She did not say a word, and with few exceptions did not appear to understand what was said to her. She apparently recognized persons and familiar things around her, but could not name them, and mostly did not seem to understand names spoken in her presence. The names of her little brothers seemed to be

entirely unrecognized by her. In the latter part of the third week of her disease her speech began to return; the first words she spoke were on May 10th. When asked if she wanted an orange, she in no way indicated that she knew what was meant; but when she saw the orange she recognized it and wanted it. On the 12th, when given milk, she struggled against it, but finally said, "I will take it." This was her first sentence. May 13th there was decided improvement; she asked for a drink in a hesitating way. When asked whether she wanted a doll, she nodded her head, and said: "Big doll, John's." May 14th she had a slight relapse; she did not speak, and did not seem to understand so well as the day before. On May 15th improvement was again decided; she could talk without difficulty, but had to stop and think before she could say anything. She then continued to improve and had no difficulty in talking. This patient had no form of paralysis, motor or sensory, but was exceedingly weak in her limbs for a long time after her recovery.

One case of paralysis which followed typhoid fever was treated by me for several months.

CASE XVIII.—PARALYSIS FOLLOWING TYPHOID FEVER.

R., six years old, was ill for nine weeks with typhoid fever. Her convalescence was tedious, but she got on her feet; and two months after recovery her mother noticed that she fell frequently, but it was not at first suspected that she was suffering from paralysis. After this weakness had been observed for about a week or two, she was carefully examined, and it was found the left leg was partially paralyzed and somewhat smaller than the right. She was then put upon massage and electricity, with tonics internally, and made considerable improvement.

She was first seen by me nearly four years after the attack of paralysis, having in the meantime been treated at intervals with electricity and massage. The left lower extremity appeared to be distinctly smaller than the right. The following measurements were taken:

From ant. sup. spinous process to patella, right,	-	32.25	inches.
" " " " " " " left,	-	32.25	"
" patella to internal malleolus, right,	-	29.5	"
" " " " " " left,	-	29.00	"
At right instep,	-	6.5	"
" left " " " " " " "	-	6.1	"
" right metacarpo-phalangeal articulation,	-	6.5	"
" left " " " " " " "	-	6.1	"

Examination of particular muscles showed that the loss of power was chiefly in the extensor proprius pollicis, extensor longus digitorum, tibialis anticus, and sartorius muscles of the left side. Reactions to both faradism and galvanism could be obtained in the muscles, however, but the responses to the current were depressed. It was particularly difficult to get a response in the tibialis anticus.

In walking, the foot was carried nearly straight, or, if anything, slightly inward; knee-jerk and muscle-jerk were present on both sides. When the treatment was stopped the muscles seemed to be gradually gaining power and their electrical properties.

The true nature of the case would seem to be somewhat doubtful. The responses to the electrical current were not typical degeneration reactions, but the case in other features did not seem like one of cerebral type, and it may be that the nerves and spinal centres, at first affected, had under time and treatment largely regenerated.

According to Gowers, limited atrophic paralysis sometimes occurs in typhoid fever, especially during convalescence, and he believes that in some cases the lesion is acute anterior poliomyelitis. As in other cases of poliomyelitis considerable amount of recovery occurs, but permanent atrophy of some muscles is left. This affection, according to him, is more frequently secondary to typhoid fever than any other acute specific disease, although he believes that many of the post-typhoid fever paralyses are due to multiple neuritis. He cites a case of Alexander's in which typhoid fever was followed by wasting of muscles with loss of electrical irritability, and of the knee-jerk, and of one vocal cord, and in which the development of the palsy was accompanied by severe pain in the legs. This patient completely recovered. He believes also that the atrophy which follows typhoid fever is local; the symptoms are neuritic rather than spinal.

Paraplegic weakness, according to Gowers, is not uncommon, especially when the disease is on the decline or during convalescence. Money has found that there is usually an excess of myotatic irritability during the disease. In other cases actual paraplegia comes on rapidly with spinal tenderness, hyperæsthesia, and other sensory manifestations, the symptoms being probably due to a slight myelitis. Even acute ascending paralysis and death have been known to occur.

Various interesting points as to pathology, diagnosis, prognosis, and treatment arise in connection with the study of a series of cases such as has been presented in this paper. To some of these attention has been directed in the body of the paper, and, in concluding, I will again briefly refer to them, and will consider also a few additional features of practical interest.

Some of these cases show the occurrence of either neuritis alone, or poliomyelitis alone, or both of these diseases conjointly, during or following affections which are universally recognized as contagious or infectious in character, namely, measles, scarlet fever, diphtheria, whooping-cough, mumps, etc. These facts suggest the idea that all cases of this type of infantile and juvenile poliomyelitis may be infectious in origin, and their well-known mode of onset would favor this view. The initial symptoms, as given by Sinkler," are fever and vomiting, diarrhoea, restlessness, and cries when moved or handled—paralysis soon following. Reasoning by analogy, it would seem that if poliomyelitis, with or without multiple neuritis, occurs after well-known infectious disorders, so, also, poliomyelitis of the usual type, ushered in by special febrile symptoms, may be infectious in nature. That this latter affection seldom occurs as an epidemic may seem to be against the view of its infectious origin, but I have found at least one record of such an epidemic in an article by Sinkler, who refers to an epidemic of infectious poliomyelitis, reported by Cordier, who saw thirteen cases between June and July, 1885, in a district containing fourteen or fifteen hundred inhabitants. The patients who survived showed the different characteristics of the affection. The fever was variable; convulsions occurred in about one-half the cases; profuse sweating was also observed; and the paralysis was present after the second or third day, in all four extremities at the same time. In some cases the neck muscles were paralyzed; in others the children were unable to nurse or cry. The gray nuclei of the ventricles were probably involved in fatal cases. In the cases which recovered paralysis did not disappear

with equal rapidity; in some there was improvement and gradual disappearance of the paralysis, which finally remained fixed in a single muscle or group of muscles, while in other cases atrophy and deformities remained.

Cordier regards the disease as having been due to an infectious agent, inspired with the air, and gives facts to sustain this position; but Sinkler is not inclined to accept the theory of infection. Bearing upon this question are a number of facts which have been recorded regarding the nature of the organic nerve lesions which occurred during or as a sequence to influenza, which included many cases of neuritis both multiple and localized, and a few of poliomyelitis and polioencephalitis.

Jacobi,¹⁶ in discussing the etiology of ordinary anterior poliomyelitis, mentioned as one almost certain cause, the presence of some poison in the blood, and gives some interesting cases in support of this view. She says, however, with reference to the theory that all cases of acute infantile paralysis are due to a specific infecting agent, that the occurrence of the spinal accidents after the ordinary infectious disease, as scarlatina and measles, should as well indicate that a specific agent proper to itself was at least not essential to its development.

From a study of the eighteen cases reported in this paper, and the brief references made to the literature of this subject, it will be seen that the lesions present in the paralyzes of infectious disease may be cerebral, spinal, or neural, or may affect at the same time several portions of the nervous system. Any of these forms of paralysis, as has been pointed out, in connection with several cases, may be classed as toxæmic, that is, due to the action of poisonous organisms, either directly or indirectly, but not necessarily giving rise to inflammation of either the nerves, the cord, or the brain. The changes in structure and the symptoms, are, in other words, dependent upon some poisonous agent, probably a ptomaine or ptomaines generated by the specific infection of the disease. In all cases this morbid agent plays its part, but in a large

percentage the pathological process does not go on to the setting up of true inflammation.

Some cerebral cases are clearly the result of hemorrhage, embolisms, etc., whatever may have been the steps which have led to the occurrence of these lesions. A few of these cases are probably forms of true encephalitis, localized or more or less diffused. In many of them, however, the functions of the brain would simply seem to have been arrested or inhibited by toxic agency.

With regard to spinal cases, it is only necessary to say, that some of them are of the type of either poliomyelitis, or a more diffused myelitis. These cases differ largely in severity and destructiveness, from cases of the mildest type with speedy recovery, to those which go on to greater or less destruction of the anterior horns.

Local, diffused, or multiple neuritis somewhat frequently occurs during or after infectious diseases, and may be associated or not with spinal or cerebral disease. Some striking examples are given in this series of cases.

The mistake must not be made by supposing that every case of paralysis, cerebral or spinal, in which pain, or even neuritis, is present in the paralyzed members, has been originally a case of concurrent neuritis and poliomyelitis, or neuritis of any kind. In a considerable percentage of cases of paraplegia, hemiplegia, etc., from spinal or cerebral lesions—hemorrhage, thrombosis, tumors, myelitis, etc.—the limbs which are the seat of the paralysis become also the seat of intense pain, both spontaneous and on handling. The neuritis in these cases is sometimes secondary and is the result of mechanical or other causes. The arm or the leg, for instance, unsupported by the muscles which usually hold it in position, is dragged partly out of the socket, and the nerves are subjected to twisting and stretching, and a traumatic neuritis is originated. I have seen several cases of this kind at the Polyclinic, and in visiting the wards of the Philadelphia Hospital.

The diagnosis of the infectious paralytic affections in children might be made to cover the consideration of all or almost all juvenile palsies. Those cases of paralysis associated with arrest of development, or active lesions of the brain, or its membranes, are not likely, except by the accident of coincidence, to be confused with the infectious paralyses. These cases have been thoroughly considered by Osler,¹⁶ Sachs, and Peterson, and others.

The infectious palsies are to be separated from numerous other paralyses and dystrophies of children, as Friedreich's ataxia, the muscular atrophies, and pseudo-hypertrophies, etc., to which attention might be first directed shortly after the occurrence of one of the infectious diseases of children. It is only necessary to bear in mind this possibility, and to make a careful examination both into the history and symptomatology for such degenerative diseases. Even locomotor ataxia of the ordinary type may develop in childhood, and the paralytic and ataxic disorders of infectious diseases have not a few symptoms in common with these cases.

The affection which has been described by Berg¹⁷ as rachitic pseudo-paraplegia must sometimes be separated. These rachitic patients are often unable to walk, or are extremely weak, but they have the general signs of rachitis in the condition of the sternum, spine, ribs, liver, etc. They may even exhibit tenderness of the muscles and bony prominences, making them thus far simulate neuritis; but close examination will show that the muscles are not really paralyzed or atrophied. The electrical reactions are normal, and these patients usually get well soon under the influence of fresh air, good food, cod-liver oil, and preparations containing iodine, such as Lugol's solution, syrup of the iodide of iron, and hydriodic acid.

Taylor¹⁸ has put on record two children in the same family suffering from pseudo-palsy of rickets, with an interesting family history. These children propelled themselves along the floor by means of their hands pressed down on either side of them, sitting and giving their bodies a forward push, they then put both hands

forward and sprang their bodies through the arch of their arms, and so really made very good speed. They all showed the superficial features of rickets; a flattened head and face, slightly curved long bones, cup-shaped chests, beaded ribs, etc. The family had been a very prolific one, usually six to seven children to each of them, and yet a score or more of these children were known to slide along the floor, just as these did, until three or four years of age, when they gradually attained the use of their legs.

With reference to the diagnosis of these cases, Taylor says there will be none of the ordinary symptoms of true palsy present, and the knee-jerk should be normal, although the knee-jerk in young children is subject not only to individual, but to accidental variations.

While discussing this question of the diagnosis of rachitic pseudo-paralyses, it might be well to note the fact that in children of the rachitic or strumous diatheses, the infectious diseases will not only give rise to paralytic disorders, but will also sometimes develop active rachitic or scrofulous affections of the bone, liver, and other tissues and organs. The following two cases, although not included in the series, are of interest in this connection:

A boy, nineteen months old, who had learned to walk, had scarlet fever with which he was ill two weeks. After that he had lost the ability to walk, but had learned to stand up by a chair, and move himself around the floor. When two years old he had whooping-cough, and again became paralyzed in both legs. When he appeared at the Polyclinic a few months after this, he was entirely helpless in both legs which were wasted, and the muscles failed to respond to faradism or galvanism. He also had a prominence in the lumbar region, such as is sometimes left after osteitis and caries. The patient was only brought a time or two to the service, and the case was not fully studied; it was probably one in which both cord and bone disease had originated in a strumous child under the influence of two infectious diseases.

Another little patient, seventeen months old, six months before coming to the clinic had scarlet fever, and two months later had measles, after which it was noted that his head was turned constantly toward the left side. He showed want of development on the right side, more marked in the arm, and marked curvature to the left in the dorso-lumbar region. His abdomen was protruding and he had an enlarged liver; his ribs were beaded and he was rachitic in general appearance; his head was twisted to the left and slightly backward. It is probable that more or less of his torticollis was present as part of his general condition before his attack of scarlet fever or measles.

Almost any form of paralysis may occur in children as the result of hysteria, and such hysterical palsies are perhaps more likely to occur after exhausting diseases. The presence of peculiarly disturbed anæsthesia, of spastic seizures or contractions, and of other hysterical symptoms, and the non-involvement of the reflexes, superficial, deep or organic, except in the way of a slight exaggeration, and the result of treatment will assist in making the diagnosis, as has been pointed out by Hun.¹⁹

Occasionally hysterical paralysis or other hysterical phenomena, are developed after infectious diseases, probably not because of any specific action of the poison of the disease, but rather owing to the general nervous weakness produced in a hysterically predisposed patient by a severe illness. A case of this kind is reported by Mills²⁰ in the "American System of Practical Medicine," and as this patient was for some time in attendance at the Polyclinic service, I will briefly refer to the case. Three months before first coming to the Polyclinic she had a sore throat with difficulty in swallowing and some regurgitation of food. By one of her medical attendants the case was regarded as diphtheritic. She developed a train of hysterical phenomena which included aphasia, a false pleuro-pneumonia, partial paralysis of the arms, numbness of her legs and feet, facial spasm, clonic torticollis and dilatation of the pupils, diplopia, blepharospasm, false luxation of the elbow, and dyspnoea.

The comments which are made on this case are sufficient to emphasize this diagnostic point, that it is important not to confuse in children or in adults, the hysterical palsies which occur after infectious diseases, with toxæmic or organic paralyses. The case was supposed by one of the attendants to be diphtheria. Subsequently she developed, over a period of many months, a train of remarkable hysterical symptoms, including in succession aphasia and pseudo-pneumonia.

"The patient's train of symptoms began with what appeared to be diphtheria. The fact that she had some real regurgitation would seem to be strong evidence that she had some form of throat paralysis following diphtheria. She was of neurotic temperament. From the age of seven until ten years she had had fits of some kind about every four weeks. Because of her sore throat and subsequent real or seeming paralytic condition, she came to the Polyclinic, where she was an object of interest and considerable attention, having been talked about and lectured upon to the classes in attendance. Whether her first symptoms were or were not hysterical, those which succeeded were demonstrably of this character. Frequently some real disease is the starting-point of a train of hysterical disorders."

The prognosis of the juvenile palsies which accompany or follow infectious diseases is of great importance. The history and character of each case must be carefully studied before a prognosis is given. While a large majority of infectious paralyses are undoubtedly curable, some are permanent or can only be partially remedied by time and treatment. The necessity of distinguishing among cases not only as to their location in the brain, spinal cord, or peripheral nerves, but also as to their nature and extent is here apparent. The cases few in number, due to central hemorrhage, embolism, etc., will be permanent or will make only partial recoveries. How far central inflammatory affections, such as encephalitis and myelitis will improve, can only be determined by

the severity of the symptoms and the lapse of time. Cases of uncomplicated neuritis are usually of hopeful prognosis, although judicious treatment is of great importance, and the paralyses may persist for a long time. The numerous cases of multiple neuritis or irregularly distributed palsies which have been so often referred to, and which for want of a better name may be simply classed as toxæmic, present a favorable prognosis, even when they are apparently of a most serious type.

The treatment of the epidemic and post-infectious paralyses of children is a subject of the greatest interest to every practicing physician who has to deal with children or with nervous affections in general. The prophylaxis of such palsies is of great importance. It is probable that in a comparatively large number of cases the development of their paralyses might be prevented by active and appropriate treatment. Usually it has been considered that no agent has any particular influence on the underlying morbid process, but this is probably not correct. The active use of remedies like the bicarbonate or citrate of potassium, mercury, and the salicylic compounds, at the right stage, and properly supported by tonics and nutrients, might perhaps do much toward preventing the occurrence of serious nervous lesions which are apparently toxic or bacterial in origin.

Special lines of treatment must, of course, depend upon the peculiarities of special cases. When a well-defined multiple or diffused neuritis is present, absolute local rest should be enforced until the signs of inflammation have subsided. The limbs should be splinted with sand-bags, and should be handled with the greatest care. The salicylates, iodides, and mercury should be used internally, but with caution. Hot douches to the limbs and dry warmth between these application will be found very useful. Generally the treatment laid down in neurological text-books should be followed. The toxic or toxæmic palsies without pain and hyperæsthesia will do best on the internal administration of drugs like

strychnine, arsenic, quinine, and iron. The presence of the rachitic or the strumous diatheses should always be taken into consideration, and if present, cod-liver oil, iodine preparations, and nutrients, with fresh air, should be ordered.

In the chronic stages of these disorders the use of faradic and galvanic electricity, massage, Swedish movements, and systematic gymnastics, after the method commonly advocated for diphtheritic and other forms of infectious paralyzes, should be persistently pursued.

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